ACUTE TOXICITY SUMMARY

HYDROGEN SELENIDE

(hydrogen selenide, selenium hydride)

CAS Registry Number: 7783-07-5

I. Acute Toxicity Summary (for a 1-hour exposure)

Inhalation reference exposure level	**5 μg/m³**
Critical effect(s)	signs of eye and respiratory irritation in guinea pigs during exposure. (Difficulty in breathing and inactivity were observed after the exposure.)
Hazard Index target(s)	Eyes; Respiratory System

II. Physical and Chemical Properties (HSDB, 1994 except as noted)

Description	gas
Molecular formula	H_2S
Molecular weight	80.98
Density	3.31 g/L @ 25°C
Boiling point	-41.3°C
Melting point	-65.73°C
Flashpoint	not applicable
Explosive limits	not applicable
Solubility	soluble in water, carbonyl chloride and carbon disulfide
Odor threshold	0.3 ppm (AIHA, 1989)
Odor description	garlic odor (AIHA, 1989)
Metabolites	trimethylselenonium (Palmer *et al*., 1970)
Conversion factor	1 ppm = 3.31 mg/m³ @ 25°C

III. Major Uses or Sources

Selenium occurs in four distinct valence forms: selenates (6+), selenite (4+), selenides (2-), and elemental (0) (Amdur *et al*., 1991). Selenite (4+) compounds and elemental selenium are believed to be of low toxicity because of their insolubility in biological media. Selenates are more acutely toxic due to their greater solubility.

The most acutely toxic selenium compound reported is hydrogen selenide (H_2Se). Hydrogen selenide is formed by the reaction of acids or water with metal selenides or by the contact of nascent hydrogen with soluble selenium compounds (Clayton and Clayton, 1982). Hydrogen selenide has no reported commercial use.

Selenium compounds are used as a decolorizing agent in the glass industry, as a vulcanizing agent in the rubber industry, in insecticides, and in photoelectric cells. Selenium compounds are also found in the toning baths used in photography and xerography. Selenium sulfide (SeS) is used in shampoos as an antidandruff agent. Up to 90% of the selenium content in ambient air is emitted during the burning of fossil fuels (Kut and Sarikaya, 1981).

The most widely used selenium compound in industry is selenium dioxide (SeO_2) (HSDB, 1994). It is produced by the oxidation of Se with nitric acid followed by evaporation or by burning Se in oxygen.

Selenium is an essential trace element in many species, including humans (Amdur *et al*., 1991). However, the dose differential between acute toxicity and chronic deficiency is slight. While the lower limit for acute oral selenium toxicity is reported to be 200 µg Se/day in humans, the "normal" oral intake is reported as 70 µg Se/day, and the oral level associated with disease due to chronic deficiency is 20 µg Se/day.

IV. Acute Toxicity to Humans

Eye, nose and throat irritation and headaches were reported by workers briefly exposed to high, but unquantitated, concentrations of selenium fume (Clinton, 1947). One worker reported delayed symptoms of sore throat and dyspnea 8-12 hours following exposure.

In a review of the literature and a report of five cases, Buchan (1947) reported that signs of acute intoxication following exposure to 0.21 ppm (0.7 mg/m³) H_2Se included irritation of the respiratory tract, severe bronchitis, bronchial pneumonia, and pulmonary edema. This report reflects occupational exposure; the exact duration of exposure was not specified. In another report, workers accidentally exposed to selenium oxide reported initial symptoms of bronchospasms, irritation of the upper respiratory passages, violent coughing, and gagging with nausea and vomiting (Wilson, 1962). Late onset symptoms observed 2 or more hours following exposure included fever, chills, headache, and dyspnea. Symptoms of bronchitis persisted for four days.

Predisposing Conditions for Selenium Toxicity

Medical: Persons with preexisting eye, skin, or respiratory conditions (including allergies) may be more sensitive to the effects of exposure to H_2Se (Reprotext, 1999).

Chemical: Persons exposed to multiple selenium compounds over time may be more sensitive to the effects of additional Se exposure (Reprotext, 1999).

V. Acute Toxicity to Laboratory Animals

The 2-hour LC_{50} in guinea pigs is 3.6 ppm (12 mg/m³) H_2Se (Dudley and Miller, 1941). No increase in mortality was observed in rabbits and guinea pigs exposed to 33 mg/m³ Se dust for 4 hours every other day for 8 days (total duration of exposure of 16 hours) (Hall *et al*., 1951). Moderate interstitial pneumonitis and congestion of the lungs was noted in both species at necropsy. A 10% mortality rate was observed in rats exposed to the same concentration of Se dust for a total of 8 hours; mild pneumonitis was noted at necropsy.

Signs of nasal and ocular irritation, including nasal discharge and pawing of the eyes and nose, were observed in guinea pigs exposed to 0.9-57 ppm (3-190 mg/m³) H_2Se for 60-minutes (Dudley and Miller, 1937). Decreased activity, marked difficulty in breathing, and decreased food intake were noted in those animals surviving the exposure. No significant increase in mortality as compared to controls was observed in guinea pigs exposed to 3 mg/m³ H_2Se for 1 hour. (Three of the 32 control animals died during the 30 day observation period following exposure while 1 of 16 animals exposed to either 3 or 4 mg/m³ H_2Se died during the observation period).

No histological changes or other signs of toxicity were observed in rats following a 1-hour exposure to 1,607, 4,499, or 8,034 ppm (7,200, 20,000, or 36,000 mg/m³) dimethylselenide vapor (equivalent to 5,200, 15,000, or 26,000 mg Se/m³) (Al-Bayati *et al*., 1992).

Microorganisms in the soil and plant products can methylate selenium to form dimethylselenide and, subsequently, dimethylselenide has been shown to be released as a vapor from acidic soil.

Rats were exposed to 2.6 mg/m³ Se^0 for 10 minutes and sacrificed 4 hours later; 57% of the Se deposited in the lungs had been absorbed into the blood (Medinsky *et al*., 1981). The single largest fraction of the excreted Se (20-28%) was found in the urine.

VI. Reproductive or Developmental Toxicity

Female Japanese rectifier workers known to be exposed to selenium reported irregular menstrual bleeding (Nagaii, 1959). The original article was not available for review and no additional information was reported in the secondary source (Friberg *et al*., 1986). No other reports of human reproductive or developmental toxicity following exposure to Se were available.

A dose-dependent increase in fetal malformations was observed following a single oral administration of 90, 100, or 110 mg/kg sodium selenate (Na_2SeO_4) to pregnant hamsters on the 8th day of gestation (Ferm *et al*., 1990). A significant decrease in fetal body weight and crown-rump length were observed following a single maternal oral dose of 110 mg/kg Na_2SeO_4. Maternal toxicity, as indicated by a significant weight loss, was observed following a single oral dose of 110 mg/kg Na_2SeO_4; approximately 30% of the dams in this group died following administration of the dose.

Dose-dependent injury to the testes of male rats was observed following a 90-day intraperitoneal administration of 2, 6, or 10 mg/day selenium dioxide (SeO_2) (Chowdhury and Venkatakrishna-Bhatt, 1983). Statistically significant decreases in relative testes weight, seminiferous tubular diameter, and Leydig cell population were observed following exposure to 6 or 10 mg SeO_2/day.

Significant testicular degeneration and testicular atrophy were observed following administration of the higher dose.

VII. Derivation of Acute Reference Exposure Level and Other Severity Levels (for a 1-hour exposure)

Reference Exposure Level (protective against mild adverse effects): 5 μg/m³

Study	Dudley and Miller, 1937; Dudley and Miller, 1941
Study population	groups of 16 guinea pigs; 32 controls
Exposure method	inhalation in a chamber
Critical effects	signs of eye and respiratory irritation, with persistent coughing after exposure, for several days.
LOAEL	0.9 ppm (3 mg/m³)
NOAEL	not observed
Exposure duration	1 hour
Extrapolated 1 hour concentration	0.9 ppm (3 mg/m³)
LOAEL uncertainty factor	6
Interspecies uncertainty factor	10
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	600
Reference Exposure Level	0.0015 ppm (0.005 mg/m³; 5 μg/m³)

Guinea pigs exposed to 0.9 ppm (3 mg/m³) H_2Se for 1 hour exhibited acute eye and nasal irritation (indicated by pawing of the nose and eyes) during the exposure and marked difficulty breathing and decreased activity following the exposure. The range of exposure concentrations was 0.9-57 ppm (3-190 mg/m³) and a 30 day observation period followed the exposure. Nearly 100% of the animals were dead within 30 days of exposure to concentrations of H_2Se of 6 ppm (20 mg/m³) and greater. No increase in mortality was observed in animals exposed to 3 or 4 mg/m³ H_2Se compared to control animals. The LOAEL for irritant effects is 0.9 ppm (3 mg/m³) H_2Se. The signs reported by the authors indicate that the irritation experience by the animals was at least moderate and may have approached a severe level.

Dudley and Miller (1941) exposed guinea pigs to hydrogen selenide for periods of 2, 4, or 8 hours. The 8-hour exposure resulted in 8/16 (50%) mortality in the animals when exposed to a concentration of 1 mg/m³. The dose-response is very steep for hydrogen selenide.

Since H_2Se is reported to be the most acutely toxic selenium compound (Amdur *et al.*, 1991), this level is considered to be protective against adverse effects from other selenium compounds as well. Use of this value for some selenium compounds will overestimate health risks. Thus, its use should be restricted to evaluating emissions of hydrogen selenide. OEHHA will continue to evaluate the literature for other selenium compounds for the development of RELs for selenium salts.

Level Protective Against Severe Adverse Effects

No recommendation is made due to the limitations of the database.

Level Protective Against Life-threatening Effects

No recommendation is made due to the limitations of the database.

NIOSH (1995) lists an IDLH of 1 mg Se/m³ based on acute toxicity data in animals. "This may be a conservative value for selenium compounds in general since it is based on sodium selenite, which is orders of magnitude more toxic than many other selenium compounds. Further, this may also be a conservative value due to the lack of relevant acute toxicity data for workers." Due to the uncertainty this value cannot be recommended.

VIII. References

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